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## DIAGNOSTIC IMPLICATIONS OF PERICARDIAL INVOLVEMENT IN CARDIOVASCULAR DISEASE

Acute myocardial infarction, dissecting aneurysm, acute idiopathic pericarditis and pulmonary embolism are characterized by chest pain, fever, leukocytosis, increased sedimentation rate, and an abnormal electrocardiogram. The criteria customarily employed in the differential diagnosis of these four diseases may be found wanting or unreliable, since the pain, electrocardiogram, and other clinical features and laboratory findings may be remarkably similar in all four conditions. The differential diagnosis, therefore, may be extremely difficult, or well nigh impossible, under certain circumstances. In cases of this sort considerable help may be obtained from the detailed observation and consideration of pericardial, pleural, and pulmonary signs.[1] The pericardial signs alone will be discussed in the present communication.

### PULMONARY EMBOLISM

Auscultatory findings which simulate those heard when there is pericardial inflammation are occasionally observed in pulmonary embolism. The friction-like sound, however, is sharply localized to the pulmonary valve area, differing in this respect from a true friction-rub of fibrinous pericarditis. Furthermore, this peculiar sound appears to be associated with the acute cor pulmonale; and, therefore, some or all of the manifestations of the latter condition are likewise observed. This is particularly true of the dilated and hyperactive pulmonary conus. The spurious friction like sound is usually ephemeral, disappearing when the dilatation and hyperactivity of the pulmonary conus subside. The close association of the two phenomena suggests a cause and effect relationship. At any rate, actual pericardial involvement does not occur; and, therefore, pericardial effusion has not been observed in pulmonary embolism.

### ACUTE IDIOPATHIC PERICARDITIS

The distinguishing features of acute idiopathic pericarditis are early, persistent, and protracted involvement of the pericardium, concomitant inflammation of the pleura, and acute cardiac dilatation. A pericardial friction rub is heard with the very onset of pain and may be audible at every subsequent examination for days or for several weeks. The cardiac silhouette may remain unaltered, or may enlarge, sometimes markedly so. Even with extreme enlargement the cardiac pulsations, though feeble, can be visualized fluoroscopically. Pericardial paracentesis may fail to yield fluid, or the amount of fluid obtainable will be incommensurate with the great increase in heart size. It is believed that this increase in heart size indicates acute cardiac dilatation.[2,3] Pleural involvement always occurs but is frequently overlooked. It may occur on either or both sides and is manifested by a pleural friction rub, progressive thickening of the pleura, or pleural effusion, which is usually small in amount and exudative in character. Pleural or pericardial paracentesis is rarely, if ever, necessary for the relief of symptoms. Sanguineous pericardial fluid has been observed, but no instance of hemothorax has come to the writer's attention.

### ACUTE MYOCARDIAL INFARCTION

The features of pericardial involvement just described contrast strikingly with those observed when pericarditis complicates acute myocardial infarction. Pericarditis occurs in about one-fifth of the cases of acute myocardial infarction and does not begin until the necrosis and inflammatory process extend to the epicardial layer of the infarcted ventricle. Since such extension usually requires at least 24 hours, evidence of pericarditis is rarely noted within one or two days of the onset of pain. Therefore, a pericardial friction rub, when present, is usually first heard on the second or third day of the coronary attack, and almost always within the first week. The rub is ephemeral, last-

ing but a few hours, or one or two days at the most; having disappeared, it does not recur. In spite of the inflammatory process, pericardial effusion large enough to be recognized by physical examination or roentgenography does not occur. Carefully controlled observations of heart size do not disclose striking changes from day to day.

## HEMORRHAGIC PERICARDITIS SECONDARY TO HYPOPROTHROMBINEMIA

The brief duration of the pericardial friction rub in acute mycardial infarction is so characteristic that any departure from the rule at once presents urgent problems for solution. Is the diagnosis of acute myocardial infarction correct? If correct, are there serious complications, such as uremia, dissecting aneurysm with hemopericardium, or hemorrhagic pericarditis secondary to hypoprothrombinemia? The last condition, because of its lethal, yet reversible potentialities, must be given very serious consideration in all patients receiving dicumarol, especially when their prothrombin activity has been unduly depressed.[4] The clinical features observed in hemorrhagic pericarditis secondary to hypoprothrombinemia are strikingly different from those occurring in uncomplicated pericarditis secondary to acute myocardial infarction, so that its recognition is not difficult. One need only remember to consider it in every patient receiving dicumarol when a pericardial friction rub persists for days, or disappears and then reappears, or first appears after the first week. Oozing of blood may continue for days, resulting in a large hemorrhagic pericardial effusion with characteristic physical and roentgenologic signs. Sudden unequivocal enlargement of the cardiac silhouette, regardless of its contour and presence or absence of pulsations, probably indicates hemorrhagic effusion, since acute cardiac dilatation does not occur in uncomplicated acute myocardial infarction. Thus, the demonstration, in patients receiving dicumarol, of pericardial effusion or of sudden increase in the dimensions of the heart suggests hemorrhagic pericarditis. Furthermore, our observations suggest that brisk hemorrhage may occur one or more times in the course of this complication, giving rise to precordial pain and the sudden appearance of the signs of cardiac tamponade. Fever and leukocytosis are also almost invariably noted.

The above clinical features suggest the diagnosis of hemorrhagic pericarditis, and are considered sufficient cause, even in the absence of more specific evidence, for the immediate inauguration of treatment. The dicumarol is omitted, and whole blood or plasma and vitamin $K_1$ or $K_1$ oxide are administered until normal prothrombin activity is restored and maintained. Pericardial paracentesis may be a life saving procedure.

## DISSECTING ANEURYSM

Pericardial involvement is common in dissecting aneurysm of the ascending aorta, and may be due to any one or a combination of three conditions. When consequent to concomitant acute myocardial infarction, which is not a rare complication in dissecting aneurysm, it is distinguished by the features described above for this disorder. On the other hand, acute fibrinous pericarditis may call attention to the existence of uremia, arising as the result of extension of the dissection into the renal arteries, or occurring for other reasons. The third cause of pericarditis in dissecting aneurysm is external rupture of the aorta into the pericardial sac. When not immediately fatal, the ensuing pericardial hemorrhage is responsible for characteristic and diagnostic signs. Blood in the pericardial sac incites an inflammatory reaction, and a pericardial friction rub therefore becomes audible. The onset of the latter coincides with the time of external rupture, and, since rupture may occur at the very beginning of dissection or hours, days, or weeks later, the rub likewise may appear with the onset of pain or hours, days, or weeks later. It may then disappear, and recur in a most irregular fashion, or once it appears, it may persist for days to weeks. As soon as external rupture occurs, blood seeps or spurts into the pericardial sac intermittently or persistently. If the patient survives this complication for days or weeks, as not infrequently happens, a surprisingly large amount of blood and fluid accumulate, giving rise to the classical signs of pericardial effusion, and in some cases, cardiac tamponade as well. When hemopericardium occurs, fever and leukocytosis are invariably noted.

## SUMMARY

A number of diagnostic implications of pericardial involvement have been described. Their careful consideration greatly enhances the possibility of correct diagnosis. Indeed, failure to evaluate properly the pericardial signs may lead to confusion and serious error in diagnosis.

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